Review

Effects of heat on performance in resistance sports in the various intensity-duration domains: review article

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Summary

Physical exercise induces an increase in body temperature that is influenced by the exercise intensity, as well as by the heat stress conditions in which it is performed. Power/velocity-duration relationship (PD-VD) shows how long an exercise can be sustained depending on the power output or the velocity output. Four intensity domains can be differentiated, which will be delimited by the lactic threshold (LT), the critical power/velocity (CP/CV) and the maximum oxygen consumption (VO_{2max}). This review aims to analyze the effects of heat stress on performance in the different intensity-duration domains, as well as to identify the main physiological mechanisms responsible. In the moderate (below LT) and hard (between LT and CP/CV) intensity domains, heat impairs the performance of exercises ranging from ~40min to over 3h, with central mechanisms and glycogen depletion being the major contributors to this fatigue. In the severe domain (above CP/CV), heat negatively affects the performance of maximum exercises ranging from ~25 to ~2 min duration, with cardiovascular and peripheral factors being the main limitations. However, in the extreme domain (above VO_{2max}), heat has been considered as a key element in achieving better performance records in maximum efforts of less than 2 min, associating these improvements with central and energy availability factors. Heat greatly influences the performance of endurance sports, accelerating task failure in those efforts longer than ~2 min, and favoring those with shorter durations. Knowing these mechanisms of action can help us to identify different strategies to reduce or take advantage of their effects during training and competition.

Key words:
Endurance. Performance. Fatigue. Hyperthermia. Physiology. Review.

Efectos del calor en el rendimiento en deportes de resistencia en los diferentes dominios de intensidad-duración: artículo de revisión

Resumen

El ejercicio físico induce un aumento de la temperatura corporal que se ve influenciado por la intensidad de este, además de por las condiciones de estrés térmico en las que se realice. La relación potencia/velocidad-duración (PD/VD) muestra cómo el tiempo que un ejercicio puede ser mantenido depende de la potencia o velocidad producida, pudiendo diferenciarse 4 dominios de intensidad que estarán delimitados por el umbral láctico (LT), la potencia/velocidad crítica (PC/VC) y el consumo máximo de oxígeno (VO_{2max}). Esta revisión tiene como objetivo analizar los efectos del estrés térmico sobre el rendimiento en los diferentes dominios de intensidad-duración, así como identificar los principales mecanismos fisiológicos responsables. En los dominios de intensidad moderado (por debajo del LT) y duro (entre LT y PC/VC), el calor perjudica el rendimiento en los ejercicios que comprenden duraciones de ~40 min hasta por encima de 3h, siendo los mecanismos centrales y la depleción del glucógeno los principales contribuyentes a esa fatiga. En el dominio severo (por encima de la PC/VC), el calor afecta negativamente al rendimiento de los ejercicios máximos que van de los ~25 a ~2 min de duración, siendo los factores cardiovasculares y periféricos los limitantes principales. Sin embargo, en el dominio extremo (por encima del VO_{2max}), el calor se ha visto como un elemento clave en la consecución de mejores registros de rendimiento en esfuerzos máximos inferiores a ~2 min de duración, debiéndose estas mejoras a factores centrales y de disponibilidad energética. El calor influye en gran medida en el rendimiento de los deportes de resistencia, acelerando el fracaso de la tarea en aquellos que tienen duraciones superiores a los ~2 min, y favoreciendo aquellos de duraciones inferiores. Conocer estos mecanismos de actuación puede ayudarnos a identificar distintas estrategias para reducir o aprovechar sus efectos durante el entrenamiento y la competición.

Palabras clave:
Resistencia. Rendimiento. Fatiga. Hipertermia. Fisiología. Revisión.

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Introduction

Resistance is defined as the time limit over which work at a determined intensity can be maintained,[1] as the energy contribution from aerobic means increases over 50% of the total as exercise extends beyond one minute.[2] Various authors subsequently defined it with greater precision arguing that, in the same way, the capability to resist both physical and psychological fatigue should be distinguished from the capacity to recover quickly from the effort.[3,4] In this way, it seems that the capability to resist a determined effort should not be associated with a minimum duration and could therefore be applied both to continuous and intermittent exercises.[5]

In resistance sports, we find 5 main physiological factors that determine performance: maximum oxygen consumption (VO_{2max}), the speed or power associated with it, energy efficiency, position (%VO_{2max}) of metabolic thresholds (VT1 and VT2), and the reserve of anaerobic speed or power (RVA/RPA).[6,7] The effects of these physiological factors on performance is represented by the power-duration (PD) or speed-duration (VD) ratio.

In particular, the hyperbolic nature of the power-duration or speed-duration curve (PD/VD) shows the ratio between the power or speed produced and the time that it can be sustained. Skinner and Mclellan[8] already classified intensities into three phases related to the physiological responses observed during exercise with progressively increasing intensity. However, Burnley and Jones[9] are currently proposing four intensity domains to explain bioenergetic responses to exercise and how they relate to task failure. This refers to the point that a participant cannot or does not want to continue a physical task.[9]

These four intensity domains are marked out by three physiological milestones (lactate threshold (LT), critical power/velocity (PC/VC) and maximum oxygen consumption (VO_{2max})), that will separate the sustainable entities for hours, even minutes or seconds: moderate intensity (power or velocity below the LT), heavy (power or speed between LT and PC/VC), severe (power or speed over the PC/VC that can be sustained until the VO_{2max} is reached), and extreme (power or velocity over VO_{2max}).[9]

The moderate intensity field includes intensities under the LT (between 50-60% of the VO_{2max} in young subjects and between 70-80% in highly-trained subjects).[10] These intensities can be sustained beyond 3 hours (e.g. ultra-marathon, trail race, road cycling or long distance triathlon), because the intensity is so low that the blood lactate concentration levels and the respiratory exchange rate (RER) remain at base levels during the stable exercise.[11] As the exercise continues over time, the incapacity to produce muscular strength due to a drop in activation of the motor neurons (central fatigue)[12] is proposed as one of the main limiting factors for ultra-resistance performance,[13–15] and bioenergetic alterations might occur in the muscle, as well as an increase in the recruitment of motor units to maintain the task in hand.[16] On the other hand, peripheral fatigue associated with these intensities is most probably caused by depletion of glycogen.[10,17] In addition, dehydration and thermal stress can also adversely affect performance and the athlete's perception of their own effort during long-duration exercise.[18,19] Although fatigue is multi-factorial in the moderate intensity domain, Burnley and Jones[9] mention that central fatigue is the main determining factor.

In turn, the heavy intensity domain includes intensities from the LT to the PC/VC (70-80% of the VO_{2max} in young subjects and 80-90% of the VO_{2max} in highly-trained subjects.10 The PC or VC reflects a critical metabolic rate from which exercise is defined in a stable metabolic status (heavy and moderate intensity) of the exercise in the metabolic instability phase (severe and extreme intensity).[20.21] This gives the hyperbolic relationship between the developed power/velocity and its sustainable time. The PC/VC is highly correlated with resistance performance, associated with the respiratory compensation point (RCP)[22] and the maximum lactate stable state (MLSS),[23] although there is quite a lot of controversy on this matter.[24–26] This review does not aim to assess the terminological and methodological differences between the PC and the MLSS (for review, see[21,27]). Therefore, we will use the terms PC or CS as the maximum metabolic stable status point that separates the heavy and severe intensity domains. The exercise in PC/VC can be sustained between 25-30 min,[10,21] and it has been estimated that elite marathon runners compete at ~96% of their critical velocity.[20] Therefore, when the LT is exceeded, tolerance for exercise is limited between ~40 min and ~3 h,[28] including tests in this domain such as a marathons, cycling time trials of around an hour and Olympic distance triathlons, among others. The characteristics of the physiological response to exercise in this domain are development of the slow component of VO_2 and an increase in the blood [lactate], that will stabilise eventually.[8] Jones *et al.*[29] observed that during exercise 10% below the PC/VC, muscular phosphocreatine (PCr) and concentrations of inorganic phosphate (Pi) and the pH attained constant values within the first 2 min of exercise and remained stable during the following 20 min. However, the slow component of the VO_2 will cause the muscular glycogen to be used as the exercise goes on, both for type I fibres and type II fibres, recruiting additional fibres necessary to maintain the intensity of the exercise, and depleting the muscular glycogen at a greater velocity.[9,16,28,30] Therefore, depletion of the musculoskeletal glycogen can be key in the fatigue processes in this domain of intensity. In this respect, Burnley, Vanhatalo and Jones[31] have also proved that central fatigue can limit performance at these intensities, and there is no single mechanism that is responsible for task failure.

The severe intensity domain includes action models that range from PC/VC to VO_{2max}. Beyond the PC/VC, the muscular metabolites (PCr and H^+), blood lactate and also the VO_2 lose homoeostasis,[32] reducing muscular efficiency, which boosts the slow component of the VO_2 to its maximum, associating task failure with achieving the VO_{2peak} and an "intolerable" muscular metabolic environment.[33,34] These work intensities above the CP/CS also recruit type II muscular fibres with low oxidative capacity, where the QO_2/VO_2 ratio (and therefore $PmvO_2$) is less than in the type I fibres.[10] Furthermore, the drop in pH means that the ventilation, and so also the respiratory frequency, increase,[35] also raising the demand on the respiratory muscles, that, out of necessity, or due to fatigue, can compromise blood flow to the active musculature,[36] which causes intramuscular metabolic stress.[37] Despite not having been tested, the reduction in the excitability of the motor neuron as the contractions progress in severe intensity exercise could contribute to central fatigue.[10] Therefore, this intensity domain considers resistance events in the range of approximately 2 to 25 min,[20] covering a very wide range of events, such as in athletics, from approximately 800 m maybe even up to 10,000 m, depending on sporting prowess and gender.

The extreme intensity domain considers all intensities over the VO_{2max}, where efforts are mainly dependent on the glycolysis and the phosphagen means. In this type of effort, high ATP production rates are fundamentally associated with developing high speed or power. The maximum rate of PCr degradation comes immediately after the start of the contraction and starts to fall after 1.3 s, while glycolysis reaches its maximum rate of ATP production after 5 s and it is maintained for several seconds before it subsequently falls.[2] Consequently, the production of force will be affected as the resynthesis and ATP use rates drop. The almost complete emptying of the PCr reserves, the gradual drop in PH, the reduction in activity of the glycolysis enzymes, and problems in the transmission and inhibition mechanisms for the motor neurons will be the factors limiting the extreme intensity performance, among many others that have yet to be determined.[2,38] Therefore, the athlete will fail the task before they can reach VO_{2max}. Athletics disciplines between 60 m and 400-800 m fall within this domain, just like some specific key moments in many resistance sport disciplines such as changes in pace, sprint finishes.

Therefore, there are many factors limiting performance that play a major role in task failure in each intensity domain. However, this review does not aim to analyse them all. The aim of this review is to analyse the acute responses from the organism to exercise in hot conditions and assess its effects on the different intensity-duration domains. It has been scientifically demonstrated that thermal stress reduces a human's capability to do exercise,[39] and it is a determining factor in certain competitions that are held each year in adverse weather conditions.[40]

Acute responses from the organism when exercising in heat

We must differentiate between the terms thermal stress and thermal tension.[41] We talk about thermal stress as environmental conditions that lead to an increase in body temperature.[42] However, thermal tension is the physiological consequence of thermal stress.[43] The interaction between the two is a complex mechanism that depends on environmental variables (temperature, humidity, wind speed, solar radiation, the clothes worn, etc.) and the type of exercise (running, cycling, swimming, etc.), individuals (aerobic condition, body size, acclimatisation status, hydration status, etc.) and intensity, duration and strategy of exercise pace.[41]

This multi-factorial interaction will cause core temperature to rise excessively over basal resting values (37ºC) for moderate intensity exercise in a cold-temperate environment (38ºC),[44] thereby accelerating the fatigue induced by hyperthermia (H) and reducing the time to task failure. Intense physical exercise can cause an increase in the core temperature (Tn) over 38ºC (H), altering the activity of the pre-frontal area of the brain (central fatigue)[45] and reducing the time to exhaustion during exercise in a warm environment.[39] Although the skin, muscle and brain temperatures are also affected, the core temperature seems to have the greatest impact on physiological thermoregulation.[46]

Cheung and Sleivert[47] conclude that there are at least two homoeostasis disturbances that affect exercise performance in H conditions, mainly affecting the Central Nervous System (CNS) and cardiovascular tension. However, Nybo *et al*.[41] propose an integrating model to understand the complexity of fatigue induced by H, that includes cardiovascular tension, central fatigue, peripheral fatigue and changes in ventilation.

Consequently, thermal stress will harm exercise performance at both high and moderate intensities, manifesting as lower power or speed production during the time trials,[48–53] reducing the time to task failure at set intensities,[54–57] or in standardised protocols such as an incremental test.[50,58,59]

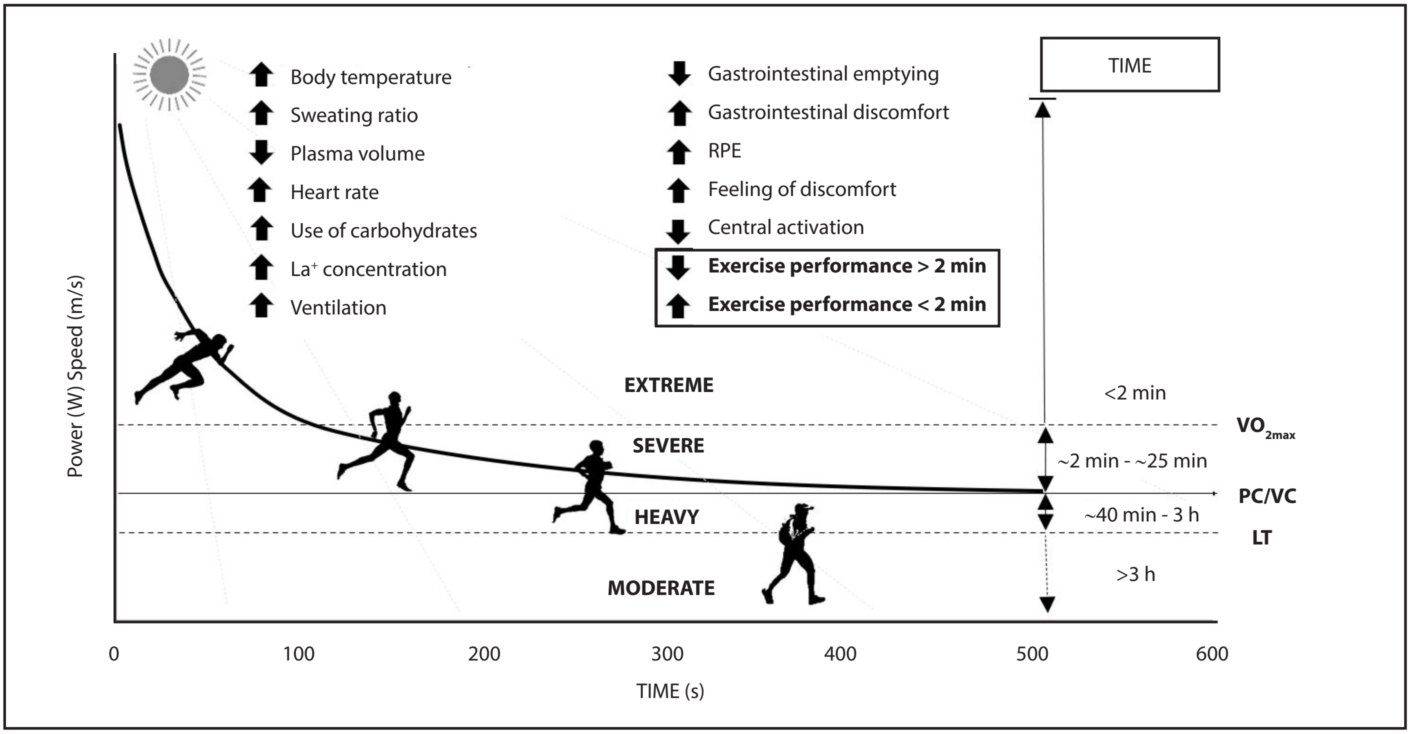
Although the fatigue induced by H is multi-factorial and many factors change in parallel or are inter-related, we will try to describe the possible causes that might induce fatigue in each intensity domain in a hot environment.

Moderate intensity domain

To the best of our knowledge, no studies have been described that compare performance in the laboratory during efforts over 3 h in control and hot conditions. However, other studies can be referenced such as Parise and Hoffman,[60] that compare data from 50 runners in a 161 km Trail event over two consecutive years, with hot conditions the first year and temperate conditions the second year, demonstrating a 7% drop in performance when competing in hot conditions. As this competition lasts around 24 h, performance was affected more for faster runners than slower ones, because they were running for a higher percentage of the race in hot conditions than runners who came further back. However, studies by Ely, Cheuvront, Roberts and Montain61 and Vihma[62] show how the lower-level runners who took more than 3 h to complete the marathon distance were more affected, as the thermal stress increased, than the runners who finished the race in a shorter time. Therefore, as these articles demonstrate, heat plays a fundamental role in developing fatigue during sub-maximal long duration races.

Factors that affect performance in the moderate intensity domain due to hyperthermia do not seem to be associated with either cardiovascular or peripheral factors.[41] Changes have barely been found in cardiac output during sub-maximal intensity exercise, due to the fact that, despite the rise in blood flow from the skin being associated with a lower systolic volume, the rise in heart rate can compensate the

Figure 1. Acute effects of the heat on exercise and the intensity-duration domains.



deterioration in cardiac filling.63 Furthermore, the increase in cardiac output will make it possible for blood to flow both to the skin and to the active skeletal muscles,[64,65] thereby helping thermoregulation and the use of oxygen to sustain the intensity of the exercise.

Thermal stress also produces changes at a metabolic level in the muscle, with a greater dependency on glycogen in hot conditions, thereby increasing blood lactate concentration levels.[59,66,67] There are several studies which state that depletion of glycogen deposits is not the main factor for task failure at sub-maximal intensities.[41,68-70] However, to the best of our knowledge, it has not been demonstrated in such long duration tests, so it will be a factor to consider in ultra-resistance competitions.

Ventilation will also be increased during the sub-maximal exercise in heat regarding the normothermia conditions,[71] but there is no mention that the respiratory musculature fatigue at these intensities causes a redistribution of the blood flow from the active skeletal musculature to the respiratory musculature,[65] so performance will not be affected by this increase in ventilation.

Therefore, the central factors seem to induce the appearance of fatigue during exercise carried out in the moderate intensity domain. The high brain temperature, the dopaminergic system activity,[72] low brain oxygen levels,[73] and feedback from the skeletal musculature[45] can affect motor activation. Furthermore, it is well documented that exercise in heat causes perceptions of greater effort than in temperate conditions.[48,72,74] The high skin temperature, associated with thermal discomfort, and the increase of ventilation, that can alter feelings of dyspnea, can also modify how exercise is performed and perceived.[37,75]

Splanchnic and gastrointestinal blood volume will also be reduced, causing the release of endotoxins that are associated with gastrointestinal discomfort, and a lower production of strength.[41,76] Gastrointestinal emptying will also be reduced by the heat,[77] accelerating dehydration and depletion of the muscular glycogen, affecting long-duration performance.

All these factors will accelerate the fatigue process in hot conditions. However, it is very complicated to separate the effects of heat from the effects of actual dehydration.

Heavy intensity domain

Ely *et al*.,[61] analysed the data from several marathons that were held at different temperatures, and they compared the results against the race records, showing a progressive drop in performance among elite marathon runners as the ambient temperature rises. Furthermore, Guy *et al*.[78] analysed the marathon times for the World Championships of the International Association of Athletics Federations (IAAF) from 1999 to 2011, and they saw a drop in performance of 3.1% and 2.7%, in men and women respectively, when the races were held in ambient temperatures >25 ˚C. Recent statistical analysis shows how, between 5ºC and 25ºC, for every 1ºC that the ambient temperature increases,

Table 1. Comparison of performance in sub-maximal aerobic exercise during time trials (TT) and time to exhaustion (TTE) in hot conditions vs. Control Temperature.

Study	N	Test	Exercise	Control	Heat	Intensity	Performance
Peiffer and Abbiss[48]	9	TT 40 km	Cycling	17°C	32°C	Self-paced	-3%
Periard *et al.*[49]	8	TT 40 km	Cycling	20°C	35°C	Self-paced	-7%
Lorenzo *et al.*[50]	12	TT 60 min	Cycling	13°C	38°C	Self-paced	-18%
Racinais *et al.*[52]	8	TT 43 km	Cycling	8°C	36°C	Self-paced	-16%
Periard and Racinais[53]	12	TT 750-Kj	Cycling	18°C	35°C	Self-paced	-14%
Periard and Racinais[86]	11	TT 750-Kj	Cycling	20°C	35°C	Self-paced	-14%
Keiser *et al.*[87]	8	TT 30 min	Cycling	18°C	38°C	Self-paced	-13%
Schlader *et al.*[88]	9	TT 30 min	Cycling	20°C	40°C	Self-paced	-21%
Romer *et al.*[89]	7	TT ~30 min	Cycling	15°C	35°C	Self-paced	-24%
VanHaitsma*et al.*[90]	20	TT 40 km	Cycling	21°C	35°C	Self-paced	-5%
Roelands *et al.*[81]	8	TT Kj 30 min	Cycling	18°C	30°C	Self-paced	-25%
Roelands *et al.*[82]	11	TT Kj 30 min	Cycling	18°C	30°C	Self-paced	-15%
Watson *et al.*[83]	8	TT Kj 30 min	Cycling	18°C	30°C	Self-paced	-30%
Supinget *et al.*[84]	10	Marathon	Running	8°C	29°C	Self-paced	-10%
Paula Viveiros *et al.*[85]	14	TT 10 km	Running	20°C	40°C	Self-paced	-21%
		TTE 90% MMP10 km		20°C	40°C	90% MMP10 km	-48%
Galloway and Maughan[54]	8	TTE	Cycling	4°C	31°C	70% VO_{2max}	-36%
				11°C	31°C	70% VO_{2max}	-44%
				21°C	31°C	70% VO_{2max}	-36%
Ftaiti *et al.*[55]	7	TTE	Cycling	22°C	35°C	60% VO_{2max}	-34%
Rowland *et al.*[56]	8	TTE	Cycling	20°C	31°C	65% VO_{2max}	-29%
Girard and Racinais[57]	11	TTE	Cycling	22°C	35°C	66% VO_{2max}	-35%
Parkin *et al.*[68]	8	TTE	Cycling	3°C	40°C	70% VO_{2max}	-64%
				20°C	40°C	70% VO_{2max}	-50%
MacDougall *et al.*[91]	6	TTE	Running	23°C	39°C	70% VO_{2max}	-36%

Km: Kilometres; Kj: Kilojoules; MMP: Personal best; VO_{2max}: Maximum Oxygen Consumption and performance reduction (%) during the tests. Dry-bulb temperature (ºC) of the control environment (temperate) and in thermal stress conditions.

marathon performance is affected by 38 s among the 100 best times, and by 20 s for the race winner.[79,80] Many studies have compared how the heat affects performance in the heavy intensity domain compared to temperate environmental conditions, distinguishing between self-paced protocols[48–50,52,53,81–90] and set intensity protocols.[54–57,68,85,91] The fixed intensity exercise protocols provide information on the time to exhaustion, while self-paced exercise protocols show the fatigue appearance process, and both protocols are based on different theories.[88] During the fixed intensity exercise, there is a progressive and not self-regulated increase in core temperature, which will lead to voluntary exhaustion of exercise when core temperature values reach ~40°C[39,45,54] or exceed them in the case of some high level athletes during competition.[92] However, during self-paced exercise, the athlete regulates the production of metabolic heat, and thereby avoids reaching this critical core temperature too soon, making it possible to finish the race (Table 1).

In this domain, the factors that produce the fatigue are the same as in the moderate intensity domain (see moderate intensity domain). The increase in dependency on carbohydrates in thermal stress conditions can accelerate the appearance of fatigue in this domain, where depletion of the muscular glycogen deposits is one of the main limiting factors, along with the central factors,[41,72,73] of the exercise between LT and PC/VC.[9]

Severe intensity domain

Several studies have seen maximum power production values fall during incremental intensity tests (VO_{2max}) in thermal stress conditions.[58,93–101] Nybo *et al.*,[41] show how VO_{2max} fell by 11% on average in 10 out of 11 studies analysed. These drops in VO_{2max} have also been seen in previously acclimatised subjects (~7%), compared with VO_{2max} in temperate conditions (21°C).[58] During timed tests or tests to exhaustion mainly

Table 2. Comparison of performance in maximum aerobic exercise during time trials (TT) and time to exhaustion (TTE) in hot conditions vs. Control Temperature.

Study	N	Test	Exercise	Control	Heat	Intensity	Performance
Periard and Racinais[86]	10	TT 15 min + 1 min 30 s max.	Cycling	18˚C	35˚C	Self-paced	-18%
Altareki *et al.*[102]	9	TT 4 km	Cycling	13˚C	35˚C	Self-paced	-2%
Ely *et al.*[103]	8	TT 15 min	Cycling	21˚C	40˚C	Self-paced	-17%
Tatterson *et al.*[104]	11	TT 30 min	Cycling	23˚C	32˚C	Self-paced	-6%
Tuckeret *et al.*[105]	10	TT 20 km	Cycling	15˚C	35˚C	Self-paced	-6%
Tyler and Sunderland[106]	9	TT 15 km	Cycling	14˚C	30˚C	Self-paced	-10%
Marino *et al.*[107]	16	TT 8 km	Cycling	15˚C	35˚C	Self-paced	-12%
Marino *et al.*[108]	12	TT 8 km	Cycling	15˚C	35˚C	Self-paced	-14%
Mitchell *et al.*[109]	11	TTE	Cycling	11˚C	37˚C	80% VO_{2max}	-48%
			Cycling	11˚C	37˚C	100% VO_{2max}	-3%

Km: Kilometres; VO_{2max}: Maximum Oxygen Consumption and performance reduction (%) during the tests. Dry-bulb temperature (ºC) of the control environment (temperate) and in thermal stress conditions.

run over the PC/VC, performance was also affected in hot conditions compared to control conditions[86,102–109] (Table 2).

During exercise in the severe intensity domain in H conditions, it seems widely accepted that cardiovascular mechanisms are responsible for the drop in VO_{2max} and performance.[41] Intense exercise is associated with high production levels of endogenous heat that, in a thermal stress environment, will compromise the capacity of the Cardiovascular System to dissipate heat to the environment,[94] thereby developing an H and compromising the capacity to diffuse arterial oxygen to the muscles that are working. The combined effect of the drop in the central blood volume,[110] and a lower diastolic filling time,[111] will cause a drop in the systolic volume and cardiac output.[63] Therefore, the muscular oxygen supply will not be sufficient for the oxygen extraction demands required by high intensity exercise, and to support thermoregulation, so performance will be affected.[41]

Therefore, the energy contribution of the anaerobic metabolism will increase to maintain the exercise intensity,[112] decreasing ATP and PCr levels in the muscles more quickly and increasing the accumulation of blood lactate and H^+, inducing peripheral fatigue.[94]

The increase in the work of the respiratory muscles during high intensity exercise will compromise the flow of blood to the musculature for the exercise due to vasoconstriction,[113,114] so it will be a relevant factor in the performance both in normothermia and hyperthermia conditions.

Furthermore, the lower oxygen delivery to the muscle and changes in the muscular metabolism will influence the inhibitory afferent feedback of the CNS, increasing the athletes' feeling of fatigue and influencing the pace,[115] limiting the development of peripheral fatigue to a critical threshold, probably to protect the organism from the extenuation and any possible damage.[116]

During the severe intensity domain, the periphery and cardiovascular factors are the main limiting factors for performance in heat, but as mentioned previously, fatigue is multi-factorial[41,47] and central fatigue can also play an important role in performance.

Extreme intensity domain

Back in 1945, Asmussen and Bøje[117] demonstrated that performance during maximum effort of 12-15 s on a static bike benefited from a greater muscular temperature, regardless of whether this was achieved by active or passive warming. Subsequently, other authors have confirmed these results on short duration maximum efforts.[118-121] However, it is not clear that performance is enhanced in severe intensity efforts in warm environment laboratory conditions. Dotan and Bar-Or[122] and Backx *et al.*[123] did not find significant differences in Wingate test performance, or several consecutive tests, between temperate and hot environments, as opposed to Ball, Burrows and Sargeant.[124] Other papers have reported greater performances in repeated short sprint protocols in thermal stress conditions.[125,126] In field conditions, Haïda *et al.*,[127] and Guy *et al.*[78] used statistical analysis of results from major international championships for sprint and middle distance races to find that there is a relationship between the best results and a greater ambient temperature. Haïda *et al.*[127] found that performances in athletics races between 100 m and 1,500 m were better in events held during the first week in July and usually coincided with important sporting events in the northern hemisphere where the average temperatures are usually high. They associate these better results with environmental conditions. Guy *et al.*[78] analysed the 6 best performances in sprint races (100-200 m) during the IAAF World Championships between 1999 and 2011, and they found that the athletes performed ~2% better in hot conditions compared to temperate conditions.

Currently, the mechanisms for causing this improved performance during short term efforts in hot conditions are not known exactly, although there are some theories. Asmussen *et al*.[117] attributed this improved performance to the fact that the increase in muscle temperature would provide a greater rate of forming cross-linkages. Gray *et al*.[120] propose a faster rate of using phosphocreatine (PCr), and a greater conduction speed for muscular fibres. For greater knowledge of heat mechanisms on sprint performance, please refer to Girard, Brocherie and Bishop.[128]

Conclusion

This review has described how thermal stress influences performance in the different intensity-duration domains described, and the physiological mechanisms that produce these variations. Analysis of competition results demonstrates how heat influences moderate intensity performance, and that it is complicated to separate the effects of heat from the effects of dehydration. There is a lot of evidence on how heat affects performance during heavy and severe intensity exercises, both in laboratory conditions and during competition. However, in the extreme intensity domain, hot conditions seem to be more favourable to develop greater power or speed than a temperate or cold environment. In sub-maximal exercises (moderate and heavy domains), central factors and dependency on muscular glycogen seem to be the main limiting factors on performance in temperate conditions and these mechanisms will be more affected as thermal stress increases, thereby accelerating the appearance of fatigue. During maximal exercises (severe domain) in hyperthermia conditions, it will be the cardiovascular and peripheral factors that cannot sustain intensity and so limit performance. Supra-maximal exercise (extreme domain) will achieve improved performance due to central factors and energy availability that increases in the heat. It is difficult to talk about isolated physiological mechanisms that determine performance in each intensity-duration domain. We talk about mechanisms that mainly influence the performance of each one, but it should be considered that many factors interact to cause the fatigue process, not just one. This review demonstrates that tests ranging from 3-4 min up to more than 3 h can benefit from cooling strategies before and during competition in hot conditions, while in races lasting <2min, cooling might compromise performance.

Conflict of interests

The authors do not declare any conflict of interests.

Bibliography

1. Bompa T. *Theory and methodology of training.* Kendall/Hunt, editor. New York; 1983.
2. Gastin P. Energy system interaction and relative contribution during maximal exercise. *Sport Med.* 2001;31:725–41.
3. Grosser M, Brüggemann P, Zintl F. *Alto rendimiento deportivo. Planificación y desarrollo.* Martínez Roca, editor. España; 1989.
4. Zintl F. *Entrenamiento de la resistencia.* Martínez Roca, editor. Barcelona; 1991.
5. Chidnok W, Dimenna FJ, Bailey SJ, Vanhatalo A, Morton RH, Wilkerson DP, *et al*. Exercise tolerance in intermittent cycling: Application of the critical power concept. *Med Sci Sports Exerc.* 2012;44:966–76.
6. Joyner MJ, Coyle EF. Endurance exercise performance: The physiology of champions. *J Physiol*. 2008;586:35–44.
7. Sanders D, Heijboer M. The anaerobic power reserve and its applicability in professional road cycling. *J Sports Sci.* 2019;37:621–9.
8. Skinner JS, McLellan TH. The transition from aerobic to anaerobic metabolism. *Res Q Exerc Sport.* 1980;51:234–48.
9. Burnley M, Jones AM. Power–duration relationship: Physiology, fatigue, and the limits of human performance. *Eur J Sport Sci.* 2018;18:1–12.
10. Poole DC, Burnley M, Vanhatalo A, Rossiter HB, Jones AM. Critical power: An important fatigue threshold in exercise physiology. Vol. 48, *Medicine and Science in Sports and Exercise.* 2016. 2320–2334 p.
11. Davies CTM, Thompson MW. Physiological responses to prolonged exercise in ultramarathon athletes. *J Appl Physiol.* 1986;61:611–7.
12. Gandevia SC, Allen GM, McKenzie DK. Central fatigue. Critical issues, quantification and practical implications. *Adv Exp Med Biol*. 1995;384:281–94.
13. Martin V, Kerhervé H, Messonnier LA, Banfi JC, Geyssant A, Bonnefoy R, *et al.* Central and peripheral contributions to neuromuscular fatigue induced by a 24-h treadmill run. *J Appl Physiol.* 2010;108:1224–33.
14. Temesi J, Rupp T, Martin V, Arnal PJ, Féasson L, Verges S, et al. Central fatigue assessed by transcranial magnetic stimulation in ultratrail running. *Med Sci Sports Exerc.* 2014;46:1166–75.
15. Lepers R, Maffiuletti NA, Rochette L, Brugniaux J, Millet GY. Neuromuscular fatigue during a long-duration cycling exercise. *J Appl Physiol.* 2002;92:1487–93.
16. Gollnick PD, Piehl K, Saltin B. Selective glycogen depletion pattern in human muscle fibres after exercise of varying intensity and at varying pedalling rates. *J Physiol.* 1974;45–57.
17. Bosch AN, Dennis SC, Noakes TD. Influence of carbohydrate ingestion on fuel substrate turnover and oxidation during prolonged exercise. *J Appl Physiol.* 1994;76:2364–72.
18. Moyen NE, Ganio MS, Wiersma LD, Kavouras SA, Gray M, McDermott B, *et al*. Hydration status affects mood state and pain sensation during ultra-endurance cycling. *J Sports Sci.* 2015;33:1962–9.
19. Hoffman MD, Stellingwerff T, Costa RJS. Considerations for ultra-endurance activities: part 2–hydration. *Res Sport Med.* 2019;27:182–94.
20. Jones AM, Vanhatalo A. The 'Critical Power' concept: Applications to sports performance with a focus on intermittent high-intensity exercise. *Sport Med*. 2017;47:65–78.
21. Jones AM, Burnley M, Black MI, Poole DC, Vanhatalo A. The maximal metabolic steady state: redefining the 'gold standard.' *Physiol Rep.* 2019;7:1–16.
22. Keir DA, Pogliaghi S, Murias JM. The respiratory compensation point and the deoxygenation break point are valid surrogates for critical power and maximum lactate steady state. *Med Sci Sports Exerc*. 2018;50:2375–8.
23. Keir DA, Paterson DH, Kowalchuk JM, Murias JM, Graham T. Using ramp-incremental VO_2 responses for constant-intensity exercise selection. *Appl Physiol Nutr Metab.* 2018;892:882–92.
24. Dekerle J, Baron B, Dupont L, Vanvelcenaher J, Pelayo P. Maximal lactate steady state, respiratory compensation threshold and critical power. *Eur J Appl Physiol*. 2003;89:281–8.
25. Dekerle J, Pelayo P, Clipet B, Depretz S, Lefevre T, Sidney M. Critical swimming speed does not represent the speed at maximal lactate steady state. *Int J Sports Med.* 2005;26:524–30.
26. Mattioni Maturana F, Keir DA, McLay K, Murias JM. Can measures of critical power precisely estimate the maximal metabolic steady-state? *Appl Physiol Nutr Metab.* 2016;41:1197–1203.
27. Broxterman RM, Craig JC, Richardson RS. The respiratory compensation point and the deoxygenation break point are not valid surrogates for critical power and maximum lactate steady state. *Med Sci Sports Exerc*. 2018;50:2379–82.
28. Coyle EF, Coggan AR, Hemmert MK, Ivy JL. Muscle glycogen utilization during prolonged strenous exercise when fed carbohydrate. *J Appl Physiol.* 1986;61:165–72.
29. Jones AM, Wilkerson DP, DiMenna F, Fulford J, Poole DC. Muscle metabolic responses to exercise above and below the "critical power" assessed using 31P-MRS. *Am J Physiol - Regul Integr Comp Physiol.* 2008;294:585–94.
30. Krustrup P, Söderlund K, Mohr M, Bangsbo J. The slow component of oxygen uptake during intense, sub-maximal exercise in man is associated with additional fibre recruitment. *Pflugers Arch Eur J Physiol.* 2004;447:855–66.
31. Burnley M, Vanhatalo A, Jones AM. Distinct profiles of neuromuscular fatigue during muscle contractions below and above the critical torque in humans. *J Appl Physiol.* 2012;113:215–23.
32. Pallarés JG, Lillo-Bevia JR, Morán-Navarro R, Cerezuela-Espejo V, Mora-Rodriguez R. Time to exhaustion during cycling is not well predicted by critical power calculations. *Appl Physiol Nutr Metab.* 2020;45:753–60.

33. Chidnok W, Dimenna FJ, Bailey SJ, Wilkerson DP, Vanhatalo A, Jones AM. Effects of pacing strategy on work done above critical power during high-intensity exercise. *Med Sci Sports Exerc*. 2013;45:1377–85.
34. Chidnok W, Fulford J, Bailey SJ, Dimenna FJ, Skiba PF, Vanhatalo A, *et al.* Muscle metabolic determinants of exercise tolerance following exhaustion: Relationship to the "critical power. *J Appl Physiol.* 2013;115:243–50.
35. Poole DC, Ward S, Gardner G, Whipp B. Metabolic and respiratory profile of the upper limit for prolonged exercise in man. *Ergonomics.* 1988;31:1265–79.
36. Taylor BJ, Romer LM. Effect of expiratory muscle fatigue on exercise tolerance and locomotor muscle fatigue in healthy humans. *J Appl Physiol*. 2008;104:1442–51.
37. Romer LM, Polkey MI. Exercise-induced respiratory muscle fatigue: Implications for performance. *J Appl Physiol.* 2008;104:879–88.
38. Girard O, Mendez-Villanueva A, Bishop D. Repeated-Sprint Ability – Part I. *Sport Med.* 2011;41:673–94.
39. Gonzàlez-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T, Nielsen B. Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol.* 1999;86:1032–9.
40. Calleja-González J, Leibar X, Arratibel I, Erauzkin J, Terrados N. Hyperthermia in top level athletes during competition. *Arch Med Del Deport.* 2009;355–9.
41. Nybo L, Rasmussen P, Sawka MN. Performance in the heat-physiological factors of importance for hyperthermia-induced fatigue. *Compr Physiol.* 2014;4:657–89.
42. Brotherhood JR. Heat stress and strain in exercise and sport. *J Sci Med Sport.* 2008;11:6–19.
43. González-alonso J, Crandall CG, Johnson JM. The cardiovascular challenge of exercising in the heat. *J Physiol.* 2008;586:45–53.
44. Nybo L. Hyperthermia and fatigue. *J Appl Physiol.* 2008;104:871–8.
45. Nybo L, Nielsen B. Hyperthermia and central fatigue during prolonged exercise in humans. *J Appl Physiol.* 2001;91:1055–60.
46. Sawka MN, Leon LR, Montain SJ, Sonna LA. Integrated physiological mechanisms of exercise performance, adaptation, and maladaptation to heat stress. *Compr Physiol.* 2011;1:1883–928.
47. Cheung SS, Sleivert GG. Multiple triggers for hyperthermic fatigue and exhaustion. *Exerc Sport Sci Rev*. 2004;32:100–6.
48. Peiffer JJ, Abbiss CR. Influence of environmental temperature on 40 km cycling time-trial performance. *Int J Sports Physiol Perform.* 2011;6:208–20.
49. Périard JD, Cramer MN, Chapman PG, Caillaud C, Thompson MW. Cardiovascular strain impairs prolonged self-paced exercise in the heat. *Exp Physiol*. 2011;96:134–44.
50. Lorenzo S, Minson CT, Babb TG, Halliwill JR. Lactate threshold predicting time-trial performance: Impact of heat and acclimation. J Appl Physiol. 2011;111:221–7.
51. Levels K, de Koning J, Broekhuijzen I, Zwaan T, Foster C, Daanen H. Effects of radiant heat exposure on pacing pattern during a 15-km cycling time trial. *J Sports Sci.* 2014;32:845–52.
52. Racinais S, Périard JD, Karlsen A, Nybo L. Effect of heat and heat acclimatization on cycling time trial performance and pacing. *Med Sci Sports Exerc.* 2014;47:601–6.
53. Périard JD, Racinais S. Performance and pacing during cycle exercise in hyperthermic and hypoxic conditions. *Med Sci Sports Exerc.* 2016;48:845–53.
54. Galloway SDR, Maughan RJ. Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Medicine and Science in Sports and Exercise*. 1997. 29:1240–9.
55. Ftaiti F, Kacem A, Jaidane N, Tabka Z, Dogui M. Changes in EEG activity before and after exhaustive exercise in sedentary women in neutral and hot environments. **Appl Ergon**. 2010;41:806–11.
56. Rowland T, Garrison A, Pober D. Determinants of endurance exercise capacity in the heat in prepubertal boys. *Int J Sports Med*. 2007;28:26–32.
57. Girard O, Racinais S. Combining heat stress and moderate hypoxia reduces cycling time to exhaustion without modifying neuromuscular fatigue characteristics. *Eur J Appl Physiol.* 2014;114:1521–32.
58. Sawka MN, Young AJ, Cadarette BS, Levine L, Pandolf KB. Influence of heat stress and acclimation on maximal aerobic power. *Eur J Appl Physiol Occup Physiol.* 1985;53:294–8.
59. Febbraio M, Snow R, Hargreaves M, Stathis C, Martin I, Carey F. Muscle metab in trained me exercise an climation tress. *J Appl Physiol*. 1994;76:589–97.
60. Parise CA, Hoffman MD. Influence of temperature and performance level on pacing a 161 km trail ultramarathon. *Int J Sports Physiol Perform*. 2011;6:243–51.
61. Ely MR, Cheuvront SN, Roberts WO, Montain SJ. Impact of weather on marathon-running performance. *Med Sci Sports Exerc*. 2007;39:487–93.
62. Vihma T. Effects of weather on the performance of marathon runners. *Int J Biometeorol.* 2010;54:297–306.
63. González-Alonso J, Mora-Rodríguez R, Coyle EF. Stroke volume during exercise: Interaction of environment and hydration. *Am J Physiol - Hear Circ Physiol*. 2000;278:321–30.
64. Savard GK, Nielsen B, Laszczynska J, Larsen BE, Saltin B. Muscle blood flow is not reduced in humans during moderate exercise and heat stress. *J Appl Physiol.* 1988;64:649–57.
65. Nielsen B, Savard G, Richter EA, Hargreaves M, Saltin B. Muscle blood flow and muscle metabolism during exercise and heat stress. *J Appl Physiol.* 1990;69:1040–6.
66. Fink WJ, Costill DL, Van Handel PJ. Leg muscle metabolism during exercise in the heat and cold. *Eur J Appl Physiol Occup Physiol*. 1975;34:183–90.
67. Young AJ, Sawka MN, Levine L, Cadarette BS, Pandolf KB. Skeletal muscle metabolism during exercise is influenced by heat acclimation. *J Appl Physiol.* 1985;59:1929–35.
68. Parkin JM, Carey MF, Zhao S, Febbraio MA. Effect of ambient temperature on human skeletal muscle metabolism during fatiguing submaximal exercise. *J Appl Physiol.* 1999;86:902–8.
69. Cheung SS. Hyperthermia and voluntary exhaustion: Integrating models and future challenges. *Appl Physiol Nutr Metab*. 2007;32:808–17.
70. Gonzalez-Alonso J, Calbet JA., Nielsen B. Metabolic and thermodynamic responses to dehydration-induced reductions in muscle blood flow in exercising humans. *J Physiol.* 1999;577–89.
71. White MD. Components and mechanisms of thermal hyperpnea. *J Appl Physiol.* 2006;101:655–63.
72. Nybo L, Secher NH. Cerebral perturbations provoked by prolonged exercise. *Prog Neurobiol.* 2004;72:223–61.
73. Rasmussen P, Nybo L, Volianitis S, Møller K, Secher NH, Gjedde A. Cerebral oxygenation is reduced during hyperthermic exercise in humans. *Acta Physiol.* 2010;199:63–70.
74. Nielsen B, Hyldig T, Bidstrup F, González-Alonso J, Christoffersen GRJ. Brain activity and fatigue during prolonged exercise in the heat. *Pflugers Arch Eur J Physiol.* 2001;442:41–8.
75. Schlader ZJ, Simmons SE, Stannard SR, Mündel T. Skin temperature as a thermal controller of exercise intensity. *Eur J Appl Physiol.* 2011;111:1631–9.
76. Jeukendrup AE, Vet-Joop K, Sturk A, Stegen JHJC, Senden J, Saris WHM, *et al.* Relationship between gastro-intestinal complaints and endotoxaemia, cytokine release and the acute-phase reaction during and after a long-distance triathlon in highly trained men. *Clin Sci*. 2000;98:47–55.
77. Neufer PD, Young AJ, Sawka MN. Gastric emptying during exercise: effects of heat stress and hypohydration. *Eur J Appl Physiol Occup Physiol.* 1989;58:433–9.
78. Guy JH, Deakin GB, Edwards AM, Miller CM, Pyne DB. Adaptation to hot environmental conditions: An exploration of the performance basis, procedures and future directions to optimise opportunities for elite athletes. *Sport Med*. 2015;45:303–11.
79. Knechtle B, Gangi S Di, Rust CA, Villiger E, Rosemann T, Nikolaidis PT. The role of weather conditions on running performance in the boston marathon from 1972 to 2018. *PLoS One*. 2019;14:1–16.
80. Nikolaidis PT, Di Gangi S, Chtourou H, Rüst CA, Rosemann T, Knechtle B. The role of environmental conditions on marathon running performance in men competing in boston marathon from 1897 to 2018. *Int J Environ Res Public Heal*th. 2019;16.
81. Roelands B, Hasegawa H, Watson P, Piacentini MF, Buyse L, De Schutter G, *et al.* The effects of acute dopamine reuptake inhibition on performance. *Med Sci Sports Exerc.* 2008;40:879–85.
82. Roelands B, Goekint M, Buyse L, Pauwels F, De Schutter G, Piacentini F, *et al*. Time trial performance in normal and high ambient temperature: Is there a role for 5-HT?. *Eur J Appl Physiol.* 2009;107:119–26.
83. Watson P, Hasegawa H, Roelands B, Piacentini MF, Looverie R, Meeusen R. Acute dopamine/noradrenaline reuptake inhibition enhances human exercise performance in warm, but not temperate conditions. *J Physiol.* 2005;565:873–83.
84. Suping Z, Guanglin M, Yanwen W, Ji L. Study of the relationships between weather conditions and the marathon race, and of meteorotropic effects on distance runners. *Int J Biometeorol.* 1992;36:63–8.
85. De Paula Viveiros J, Amorim FT, Alves MNM, Passos RLF, Meyer F. Run performance of middle-aged and young adult runners in the heat. *Int J Sports Med*. 2012;33:211–7.
86. Périard JD, Racinais S. Self-paced exercise in hot and cool conditions is associated with the maintenance of %VO2peak within a narrow range. *J Appl Physiol.* 2015;118:1258–65.
87. Keiser S, Flück D, Hüppin F, Stravs A, Hilty MP, Lundby C. Heat training increases exercise capacity in hot but not in temperate conditions: A mechanistic counter-balanced cross-over study. *Am J Physiol - Hear Circ Physiol.* 2015;309:H750–61.
88. Schlader ZJ, Stannard SR, Mündel T. Is peak oxygen uptake a determinant of moderate duration self-paced exercise performance in the heat? *Appl Physiol Nutr Metab.* 2011;36:863–72.
89. Romer LM, Bridge MW, McConnell AK, Jones DA. Influence of environmental temperature on exercise-induced inspiratory muscle fatigue. *Eur J Appl Physiol.* 2004;91:656–63.
90. VanHaitsma TA, Light AR, Light KC, Hughen RW, Yenchik S, White AT. Fatigue sensation and gene expression in trained cyclists following a 40 km time trial in the heat. *Eur J*

Appl Physiol. 2016;116:541–52.

91. MacDougall JD, Reddan WG, Layton CR, Dempsey JA. Effects of metabolic hyperthermia on performance during heavy prolonged exercise. *J Appl Physiol.* 1974;36:538–44.
92. Maron MB, Wagner JA, Horvath SM. Thermoregulatory responses during competitive marathon running. *J Appl Physiol Respir Environ Exerc Physiol.* 1977;42:909–14.
93. Arngrímsson SÁ, Stewart DJ, Borrani F, Skinner KA, Cureton KJ. Relation of heart rate to percent VO2 peak during submaximal exercise in the heat. *J Appl Physiol.* 2003;94:1162–8.
94. González-Alonso J, Calbet JAL. Reductions in systemic and skeletal muscle blood flow and oxygen delivery limit maximal aerobic capacity in humans. *Circulation*. 2003;107:824–30.
95. Klausen K, Dill DB, Phillips EE, McGregor D. Metabolic reactions to work in the desert. *J Appl Physiol.* 1967;22:292–6.
96. Lafrenz AJ, Wingo JE, Ganio MS, Cureton KJ. Effect of ambient temperature on cardiovascular drift and maximal oxygen uptake. *Med Sci Sports Exerc*. 2008;40:1065–71.
97. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. *J Appl Physiol*. 2010;109:1140–7.
98. Nybo L, Jensen T, Nielsen B, González-Alonso J. Effects of marked hyperthermia with and without dehydration on VO_2 kinetics during intense exercise. *J Appl Physiol.* 2001;90:1057–64.
99. Pirnay F, Deroanne R, Petit JM. Maximal oxygen consumption in a hot environment. *J Appl Physiol.* 1970;28:642–5.
100. Rowell LB, Brengelmann GL, Murray JA, Kraning KK, Kusumi F. Human metabolic responses to hyperthermia during mild to maximal exercise. *J Appl Physiol.* 1969;26:395–402.
101. Wingo JE, Lafrenz AJ, Ganio MS, Edwards GL, Cureton KJ. Cardiovascular drift is related to reduced maximal oxygen uptake during heat stress. *Med Sci Sports Exerc*. 2005;37:248–55.
102. Altareki N, Drust B, Atkinson G, Cable T, Gregson W. Effects of environmental heat stress (35°C) with simulated air movement on the thermoregulatory responses during a 4-km cycling time trial. *Int J Sports Med.* 2009;30:9–15.
103. Ely BR, Cheuvront SN, Kenefick RW, Sawka MN. Aerobic performance is degraded, despite modest hyperthermia, in hot environments. *Med Sci Sports Exerc*. 2010;42:135–41.
104. Tatterson AJ, Hahn AG, Martin DT, Febbraio MA, Movement H. Effects of heat stress on physiological responses and exercise performance in elite cyclists. *J Sci Med Sport*. 2000;3:186–93.
105. Tucker R, Rauch L, Harley YX, Noakes TD. Impaired exercise performance in the heat is associated with an anticipatoiy reduction in skeletal muscle recruitment. *Pflugers Arch Eur J Physiol.* 2004;448:422–30.
106. Tyler C, Sunderland C. The effect of ambient temperature on the reliability of a preloaded treadmill time-trial. *Int J Sports Med*. 2008;29:812–6.
107. Marino FE, Mbambo Z, Kortekaas E, Wilson G, Lambert MI, Noakes TD, *et al*. Advantages of smaller body mass during distance running in warm, humid environments. *Pflugers Arch Eur J Physiol.* 2000;441:359–67.
108. Marino FE, Lambert MI, Noakes TD. Superior performance of African runners in warm humid but not in cool environmental conditions. *J Appl Physiol.* 2004;96:124–30.
109. Mitchell JB, Rogers MM, Basset JT, Hubing KA. Fatigue during high-intensity endurance exercise: the interaction between metabolic factors and thermal stress. *J strength Cond Res.* 2014;28:1906–14.
110. Rowell LB, Marx HJ, Bruce RA, Conn RD, Kusumi F. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J Clin Invest*. 1966;45:1801–16.
111. Fritzsche RG, Switzer TW, Hodgkinson BJ, Coyle EF. Stroke volume decline during prolonged exercise is influenced by the increase in heart rate. *J Appl Physiol.* 1999;86:799–805.
112. Dimri GP, Malhotra MS, Sen Gupta J, Sampath Kumar T, Arora BS. Alterations in aerobic-anaerobic proportions of metabolism during work in heat. *Eur J Appl Physiol Occup Physiol.* 1980;45:43–50.
113. Harms CA, Babcock MA, McClaran SR, Pegelow DF, Nickele GA, Nelson WB, *et al*. Respiratory muscle work compromises leg blood flow during maximal exercise. *J Appl Physiol.* 1997;82:1573–83.
114. Harms CA, Wetter TJ, McClaran SR, Pegelow DF, Nickele GA, Nelson WB, *et al*. Effects of respiratory muscle work on cardiac output and its distribution during maximal exercise. *J Appl Physiol.* 1998;85:609–18.
115. Amann M. Central and peripheral fatigue: Interaction during cycling exercise in humans. *Med Sci Sports Exerc.* 2011;43:2039–45.
116. Amann M. Significance of Group III and IV muscle afferents for the endurance exercising human. *Clin Exp Pharmacol Physiol.* 2012;39:831–5.
117. Asmussen E, Bøje O. Body temperature and capacity for work. *Acta Physiol Scand.* 1945;10:1–22.
118. Sargeant AJ. Effect of muscle temperature on leg extension force and short-term power output in humans. *Eur J Appl Physiol Occup Physiol.* 1987;56:693–8.
119. Linnane DM, Bracken RM, Brooks S, Cox VM, Ball D. Effects of hyperthermia on the metabolic responses to repeated high-intensity exercise. *Eur J Appl Physiol.* 2004;93:159–66.
120. Gray SR, De Vito G, Nimmo MA, Farina D, Ferguson RA. Skeletal muscle ATP turnover and muscle fiber conduction velocity are elevated at higher muscle temperatures during maximal power output development in humans. *Am J Physiol - Regul Integr Comp Physiol.* 2006;290:376–83.
121. Yaicharoen P, Wallman K, Bishop D, Morton A. The effect of warm up on single and intermittent-sprint performance. *J Sports Sci.* 2012;30:833–40.
122. Dotan R, Bar-Or O. Climatic heat stress and performance in the Wingate Anaerobic Test. *Eur J Appl Physiol Occup Physiol.* 1980;44:237–43.
123. Backx K, Mc Naughton L, Crickmore L, Palmer G, Carlisle A. Effects of differing heat and humidity on the performance and recovery from multiple high intensity, intermittent exercise bouts. *Int J Sports Med.* 2000;21:400–5.
124. Ball D, Burrows C, Sargeant AJ. Human power output during repeated sprint cycle exercise: The influence of thermal stress. *Eur J Appl Physiol Occup Physiol.* 1999;79:360–6.
125. Falk B, Radom-Isaac S, Hoffmann JR, Wang Y, Yarom Y, Magazanik A, *et al*. The effect of heat exposure on performance of and recovery from high-intensity, intermittent exercise. *Int J Sports Med*. 1998;19:1–6.
126. Girard O, Bishop DJ, Racinais S. Hot conditions improve power output during repeated cycling sprints without modifying neuromuscular fatigue characteristics. *Eur J Appl Physiol.* 2013;113:359–69.
127. Haïda A, Dor F, Guillaume M, Quinquis L, Marc A, Marquet LA, *et al*. Environment and scheduling effects on sprint and middle distance running performances. *PLoS One.* 2013;8:1–7.
128. Girard O, Brocherie F, Bishop DJ. Sprint performance under heat stress: A review. *Scand J Med Sci Sport.* 2015;25:79–89.